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Sensory processing deficiencies in patients with borderline personality disorder who experience auditory verbal hallucinations

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ABSTRACT

Auditory verbal hallucinations (AVH) are common in patients with borderline personality disorder (BPD). We examined two candidate mechanisms of AVH in patients with BPD, suggested to underlie sensory processing systems that contribute to psychotic symptoms in patients with schizophrenia; sensory gating (P50 ratio and P50 difference) and change detection (mismatch negativity; MMN). Via electroencephalographic recordings P50 amplitude, P50 ratio, P50 difference and MMN amplitude were compared between 23 borderline patients *with* and 25 *without* AVH, and 26 healthy controls. Borderline patients *with* AVH had a significantly lower P50 difference compared with healthy controls, whereas no difference was found between borderline patients *without* AVH and healthy controls. The groups did not differ on MMN amplitude.

The impaired sensory gating in patients with borderline personality disorder who experience AVH implies that P50 sensory gating deficiencies may underlie psychotic vulnerability in this specific patient group. Patients with borderline personality disorder with or without AVH did not have problems with auditory change detection. This may explain why they are spared from the poor outcome associated with negative symptoms and symptoms of disorganization in patients with chronic schizophrenia.

1. Introduction

AVH also frequently occur in patients with borderline personality disorder (BPD) (Kingdon et al., 2010; Niemantsverdriet et al., 2017; Pearse et al., 2014; Yee et al., 2005), with a point prevalence of 21% (Niemantsverdriet et al., 2017). Among these patients, associations have been reported between the presence and severity of AVH and both suicidality and hospitalization (Slotema et al., 2017). In patients with BPD, AVH are phenomenologically similar to those in patients with schizophrenia and cause equal amounts of distress (Kingdon et al., 2010; Slotema et al., 2012; Tschoeke et al., 2014). This, and the knowledge that both schizophrenia and borderline personality disorder are often associated with childhood trauma, offers the possibility that there may be a shared underlying mechanism of AVH in these two (McCarthy-Jones and Longden, 2015; Niemantsverdriet et al., 2017). To date however, no studies have investigated the neurocognitive or neurobiological mechanisms of AVH in patients with BPD to test this hypothesis.

In the research field of schizophrenia spectrum disorders, sensory processing deficiencies have been mentioned to underlie psychotic symptoms, such as hallucinations. Javitt & Freedman (Javitt and Freedman, 2015) explain in their review that these deficits in processing auditory (or visual) stimuli may lead to psychotic symptoms in two ways: i) neuropsychologically; a failure to register basic sensory information correctly makes poor decisions about it inevitable, and ii) neurobiologically; the same neuronal mechanisms that register the information are utilized throughout the brain, so that deficits in neuronal mechanisms detected in sensory areas are likely to be present also in regions that have more complex executive functions.

The electroencephalographic (EEG) P50 gating is a widely used measure for sensory gating and is reported to be reduced in patients with schizophrenia (Bramon et al., 2004; de Wilde et al., 2007a, b; Patterson et al., 2008). A second measurement of sensory processing is change detection in auditory stimulation, as measured by mismatch negativity (MMN) of the EEG, which is also known to be affected in patients with schizophrenia (Erickson et al., 2016; Umbricht and Krljes,

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2005).

1.1. P50

P50 sensory gating reflects a basic ability of a person to inhibit one's response to the second of a pair of stimuli, hence the ability to filter out irrelevant sensory information. In P50 sensory gating, auditory event-related potentials (ERPs) are measured via EEG recordings, using a double-click paradigm. Sensory gating occurs when the amplitude of the P50 to the second click (S2) is suppressed compared to the amplitude elicited by the first click (S1). Sensory gating can be exhibited with the aid of the P50 ratio (the quotient of the P50 amplitude elicited by S2 divided by the P50 amplitude elicited by S1; S2/S1) and the P50 difference (the difference between the P50 amplitude elicited by S1 and the P50 amplitude elicited by S2; S1-S2).

Meta-analyses have demonstrated significantly larger P50 ratios compared to controls, in patients with schizophrenia (Bramon et al., 2004; de Wilde et al., 2007a; Patterson et al., 2008) and their relatives (de Wilde et al., 2007b; Earls et al., 2016) indicating that P50 sensory gating seems to be an important candidate endophenotype for schizophrenia.

A review of studies on the P50 ratio (Potter et al., 2006), revealed a relationship between the P50 ratio and sustained attention and vigilance. In addition, Smith et al. found that more effective P50 sensory gating predicted better scores on attention tests (Smith et al., 2010) and recently Hamilton et al. demonstrated covariations between P50 suppression deficiency and attention difficulties, poorer working memory, and reduced processing speed (Hamilton et al., 2018b). Three studies focused specifically on the association between P50 sensory gating deficiencies and AVH in patients with schizophrenia. These latter studies revealed significantly higher P50 ratios (i.e. more deficient sensory gating) when AVH were present (state-dependent) (Thoma et al., 2017), as well as a correlation between the severity of AVH and more deficient P50 gating scores (Faugere et al., 2016; Smith et al., 2013). The finding that P50 sensory gating deficiency is also found in patients with bipolar disorder, mainly in patients with a history of psychosis, gave rise to the hypothesis that deficient P50 gating is a more general indicator of psychotic vulnerability (Cabranes et al., 2013; Olincy and Martin, 2005; Sanchez-Morla et al., 2008; Schulze et al., 2007). This finding is in line with conclusions drawn from studies reporting reduced P50 sensory gating in patients with schizotypal personality disorder, i.e. that an abnormal P50 ratio is a neurocognitive deficit across the psychosis *spectrum* (Cadenhead et al., 2000, 2002; Hazlett et al., 2015).

Despite the widespread use of the P50 ratio, it is suggested that the P50 difference may be a more reliable measurement of P50 sensory gating. Following studies reporting low test-retest reliability coefficients for the P50 ratio, Smith et al. showed that the moderate correlation between P50 amplitudes elicited by S1 and S2 and the greater variability of P50 amplitudes elicited by S2 relative to the variability of P50 amplitudes elicited by S1, are the psychometric reasons behind the relatively less reliable measurement of P50 sensory gating via the S2/S1 ratio compared to the S1-S2 difference(Smith et al., 1994). In two heritability studies, both Anokhin et al. (Anokhin et al., 2007) and Greenwood et al. (Greenwood et al., 2007) found that the P50 difference exhibited higher heritability than the P50 ratio in both the general population and in patients with schizophrenia. Since the present study is the first P50 sensory gating study among patients with BPD who experience AVH, both of these latter measures (i.e. the P50 ratio and P50 difference) were applied.

1.2. Mismatch negativity

Mismatch negativity is the automatically and pre-attentively generated negative component of the ERP to any change in auditory stimulation (Naatanen et al., 2007). MMN requires memory of a sequence and detection of auditory variation (e.g. duration, frequency, location) in the stimulus. A decreased MMN amplitude suggests a deficiency in either the short-term memory storage of auditory information, or in the registration of a change in auditory stimulation. MMN impairment is a robust finding in studies focusing on sensory processing deficiencies in patients with chronic schizophrenia (Erickson et al., 2016; Umbricht and Krljes, 2005) and was found to be associated with the presence and severity of AVH (Ford et al., 2012), as well as with negative symptoms (Umbricht and Krljes, 2005) and worse functional outcome (Green et al., 2009; Hamilton et al., 2018a).

MMN deficiencies are not limited to patients with a diagnosis of schizophrenia, but have been found across the psychosis spectrum (Randeniya et al., 2018). Individuals with a high risk state for psychosis (ultra-high-risk; UHR) show consistent MMN deficiencies when studied with duration auditory variation. When the change in auditory stimulation was based on frequency however, both intact and deficient MMN have been found (Randeniya et al., 2018). When compared to healthy controls, patients with schizotypal personality disorder displayed reduced frequency MMN amplitude in a study by (Niznikiewicz et al., 2009). In a comparison study of patients with paranoid and schizotypal disorder however, Liu et al. found MMN amplitude similar to that of healthy controls in the first group, and a *higher* MMN amplitude in the second group, in response to frequency deviant tones (Liu et al., 2007). Accumulating research also shows that patients with bipolar disorder, albeit to a lesser degree than patients with schizophrenia, demonstrate MMN deficiencies (Hermens et al., 2018).

1.3. ERPs in BPD

Although the processing of auditory stimuli by means of ERPs has been investigated in patients with BPD, no study so far focused on psychotic symptoms such as AVH. For example, Grootens et al. examined P50 sensory gating in patients with BPD and found *increased* sensory gating in this patient group as compared to healthy controls due to a higher amplitude elicited by S1 (Grootens et al., 2008). The authors suggest that patients with BPD might have an increased physiological predisposition to respond to new stimuli and that their 'compensatory' gating mechanism seems even more efficient compared with healthy controls.

He et al. investigated MMN in patients with treatment-resistant depression compared to i) patients with treatment-resistant depression and comorbid BPD, ii) patients with BPD only, and iii) healthy controls (He et al., 2010). Patients with treatment-resistant depression displayed a higher MMN amplitude compared to the other groups, whereas for patients with BPD the MMN amplitudes did not differ from those in healthy controls.

The present study investigated whether patients with BPD who experience AVH have similar sensory processing deficiencies as observed in patients with schizophrenia. Our hypothesis was that patients with BPD *and* AVH would demonstrate a deficiency in the P50 ratio and difference and a deficiency in MMN amplitude compared to patients with BPD *without* AVH and also compared to healthy controls.

2. Methods

2.1. Participants

For participation the inclusion criteria were age ≥ 18 years and sufficient mastery of the Dutch language. For patients with BPD, the additional inclusion criteria were i) a diagnosis of BPD assessed with the Structured Clinical Interview for DSM-IV-TR (SCID II) (First et al., 1997); ii) no comorbid DSM-IV diagnosis of schizophrenia or schizoaffective disorder according to the Comprehensive Assessment of Symptoms and History (CASH) (Andreasen et al., 1992) or the MINI-International Neuropsychiatric Interview (MINI PLUS 2000) (Sheehan et al., 1998); and iii) no substance abuse (defined as alcohol consumption of ≥ 3 units/day, daily cannabis use, or use of hard drugs

≥1 time/month). AVH were defined as present when they occurred at least once per week, as established with the frequency item of the AVH-related subscale of the Psychotic Symptom Rating Scale (PSYRATS) (Haddock et al., 1999). Healthy controls consisted of participants with i) no presence or history of psychiatric illness as assessed by the MINI PLUS 2000, ii) no present use of psychotropic medication, and iii) the absence of AVH.

Participants were enrolled into this study between May 2009 and August 2016 from the Outpatient Department for Personality Disorders at Parnassia Psychiatric Institute (The Hague, the Netherlands) and Rudolf Magnus Institute of Neuroscience, University Medical Center Utrecht (the Netherlands). Patients were recruited either directly via posters in the waiting rooms, or via their therapist who informed them about the inclusion criteria for and content of this study.

The study was performed in accordance with relevant guidelines and regulations, and registered and approved by the National Medical Ethical Committee (NL1371209706). Written informed consent was obtained from all participants. Each participant received 35 euro for their participation.

A total of 23 patients with BPD *and* AVH (BPD + AVH), 26 patients with BPD *without* AVH (BPD) and 25 healthy controls participated in the study. However, because errors occurred in the MMN recordings of three participants, this resulted in measurements for MMN in: 21 patients with BPD + AVH, 25 patients with BPD and 25 healthy controls.

2.2. Stimuli and procedure

The recordings took place at the Behavioural Laboratory of Erasmus University Rotterdam. Participants were seated in a comfortable armchair in a sound-attenuated room with dimmed lights, where the elastic electrode head caps and electrodes were attached. Auditory stimuli were presented through headphones.

The P50 stimuli consisted of two identical clicks: a conditioning stimulus (S1) and a test stimulus (S2) with a fixed interstimulus interval of 500 msec. The interval between the pairs of clicks varied randomly between 8 and 12 s. Each participant received 60 trials. Participants were instructed to count the clicks in silence and were asked to report the number of clicks after finalization of the task. This was done to exclude severe hearing deficits and assure a consistent level of engagement during the task.

For MMN recordings participants were presented with 80 dB 500 Hz "frequent" stimuli with a duration of 55 ms, with a rise and fall time of 5 ms. At random times, an "infrequent" deviant 750 Hz tone was presented. In total, 700 stimuli were presented with a probability of occurrence of 0.85 for the frequent stimulus and 0.15 for the infrequent stimulus. Every 1200 ms, a stimulus was presented. Participants were instructed to ignore the tones and to read a text from a magazine.

2.3. EEG recording and signal processing

The EEG was recorded with a BIOSEMI Active-Two amplifier system, from 64 Ag/AgCl active electrodes mounted in an elastic cap. Additional electrodes were placed at the two outer canthi of the eyes and infraorbital and supraorbital regions for the detection of eye blinks and eye movement artifacts (electrooculography; EOG). Mastoid electrodes were placed for off-line re-referencing.

The EEG and EOG signals were analyzed with Brain Vision Analyzer 2 (Brain-Products GmbH, Munich, Germany). All signals were digitized with a sample rate of 512 Hz and 24-bit A/D conversion. Data were off-line re-referenced to the mastoid electrodes. Furthermore, EEG and EOG activity was off-line filtered (bandpass 10–50 Hz (Jerger et al., 1992; White and Yee, 1997; Yee et al., 1998); phase shift-free Butterworth filters; 24 dB/octave slope). Epochs including an EEG or EOG change exceeding ± 50 μV were omitted from the averaging.

The auditory ERP waveforms had to meet the following criteria. The mean 100 ms pre-stimulus period served as baseline. The P50 was identified as the largest positive peak between 50 and 100 ms post stimulus. The P50 ratio was calculated as the quotient of the P50 amplitude elicited by S2 divided by the P50 amplitude elicited by S1 (S2/S1), the P50 difference was calculated as the difference between the P50 amplitude elicited by S1 and the P50 amplitude elicited by S2 (S1-S2). Outcomes of a high P50 ratio and a low P50 difference represent impaired sensory gating. Consistent with previous studies, waveform measurements were made at Cz for P50 (Hamilton et al., 2018b; Smith et al., 2010, 2013; Thoma et al., 2017) and where the largest responses were obtained (Fz and Cz for MMN) (Naatanen et al., 2004). The MMN was obtained by subtracting the standard stimulus ERP (occurring at the 140–170 ms post-stimulus period) from the deviant stimulus (Naatanen et al., 2004).

2.4. Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 23.0 (Armonk, NY: IBM Corp). Participant characteristics were compared using one-way ANOVA (for age) and chi-square analyses (for gender, education and medication). Because of a non-normal distribution in the dependent variables (P50 amplitudes, P50 ratio and P50 difference), Kruskall–Wallis non-parametric tests were used, followed by Mann–Whitney post-hoc testing. The MMN amplitudes were compared with one-way ANOVA. A p-value of 0.05 was considered significant, with Bonferroni correction for multiple comparisons.

3. Results

3.1. Participant characteristics

Participant characteristics are presented in Table 1. The three groups did not differ with regard to age, gender and education. The groups BPD + AVH and BPD, did not differ with regard to medication use.

3.2. ERP data

Fig. 1 presents the averaged waveforms for the P50 amplitude elicited by S1 and S2. No differences could be revealed between the

Table 1
Characteristics of the three patient groups.

	BPD + AVH (n = 23)	BPD (n = 26)	HC (n = 25)	p
Age in years, mean (SD)	41.09 (13.01)	35.88 (9.69)	36.00 (12.75)	0.23
Female gender, n (%)	19 (82.6)	23 (88.5)	21 (84.0)	0.85[a]
Education, n (%)				
Primary	4 (17.4)	2 (7.7)	1 (4.0)	0.28[a]
Vocational	11 (47.8)	15 (57.7)	9 (36.0)	0.33
Selective secondary	1 (4.3)	2 (7.7)	6 (24.0)	0.11[a]
Bachelor degree or higher	5 (21.7)	4 (15.4)	9 (36.0)	0.24
Medication, n (%)				
Typical AP	4 (17.4)	1 (4.3)	–	0.35[a]
Atypical AP	4 (17.4)	7 (30.4)	–	0.30
Hypnotics	8 (34.8)	6 (25.0)	–	0.46
Antidepressants	13 (59.1)	12 (52.2)	–	0.64
Mood stabilizers	2 (8.7)	0 (0.0)	–	0.23[a]

Abbreviations: AP = antipsychotic, BPD + AVH = patients with borderline personality disorder *and* auditory verbal hallucinations, BPD = patients with borderline personality disorder *without* auditory verbal hallucinations, HC = healthy controls.
Group differences were analyzed with one-way ANOVA for age and with chi-square analysis for gender, education and medication.
[a] Fisher's exact test was used because of expected small frequencies.

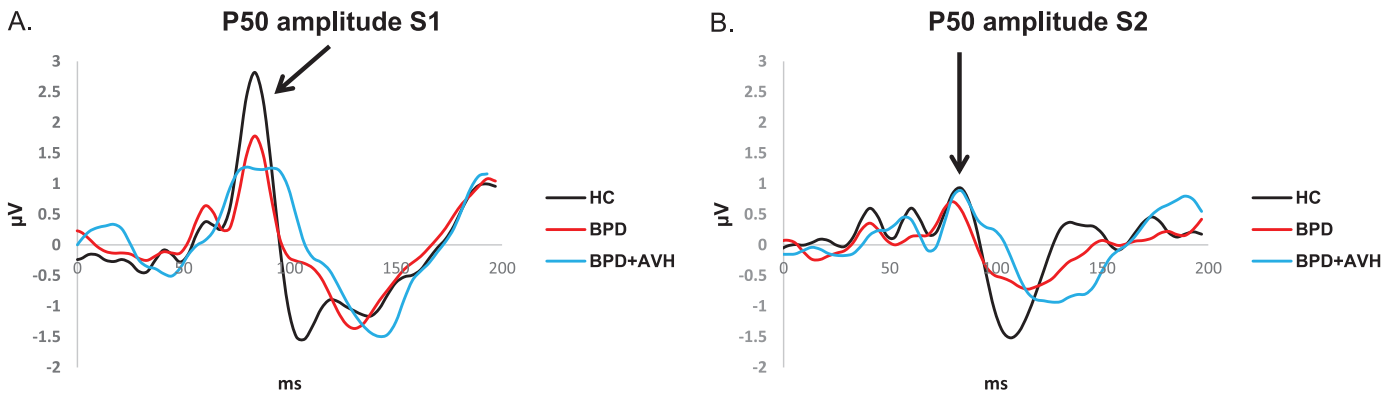

Fig. 1. Averaged waveforms for the P50 amplitude elicited by (A) S1 and by (B) S2. The lines indicate the mean values in electrode position Cz. Abbreviations: BPD + AVH = patients with borderline personality disorder *and* auditory verbal hallucinations, BPD = patients with borderline personality disorder *without* auditory verbal hallucinations, HC = healthy controls, S1 = first stimulus, S2 = second stimulus.

Table 2
Data on the P50: median (range) and test statistic.

	Median (range)					Post-hoc								
						1 vs. 2			1 vs. 3			2 vs. 3		
	1. BPD + AVH (n = 23)	2. BPD (n = 26)	3. HC (n = 25)	$H(2)$	p	Z	p	effect size (r)	Z	p	effect size (r)	Z	p	effect size (r)
P50 at Cz-site														
S1 amplitude (μV)	1.89 (−1.20 - 7.29)	2.63 (−1.10 - 5.80)	3.21 (−0.26 - 7.84)	5.63	0.06	–		–	–		–	–		–
S2 amplitude (μV)	1.54 (−0.59 - 7.93)	1.21 (−0.70 - 3.42)	1.86 (−0.52 - 3.29)	1.70	0.43	–		–	–		–	–		–
Ratio (S2/S1)	0.72 (−3.22 - 13.78)	0.38 (−2.23 - 4.03)	0.44 (−2.47 - 4.14)	3.05	0.22	–		–	–		–	–		–
Difference (S1-S2; μV)	0.53 (−5.73 - 5.21)	1.62 (−3.56 - 3.62)	1.62 (−2.03 - 5.60)	8.01	0.02	−2.30	0.02[a]	0.33	−2.53	0.01[b]	0.36	−0.70	0.49	0.11

[a] No longer significant after Bonferroni correction. [b] Remained significant after Bonferroni correction. Abbreviations: BPD + AVH = patients with borderline personality disorder *and* auditory verbal hallucinations, BPD = patients with borderline personality disorder *without* auditory verbal hallucinations, HC = healthy controls.

groups in the P50 amplitude (elicited by S2) and the P50 ratio (Table 2, Fig. 2). However, there was a trend between the groups towards a difference in the P50 amplitude elicited by S1 ($H(2) = 5.63$, $p = 0.06$). There was a significant difference between the groups in the P50 difference (S2-S1) ($H(2) = 8.01$, $p = 0.02$). The P50 difference was smaller for the BPD + AVH group compared to healthy controls (median 0.53 vs. 1.62, $Z = -2.53$, $p = 0.01$) with a moderate effect size ($r = 0.36$) (Cohen, 1992). The P50 difference was also smaller for the group BPD + AVH compared to the BPD group (median 0.53 vs. 1.62, $Z = -2.30$, $p = 0.02$). However, the latter difference did not remain significant after Bonferroni correction.

All groups displayed MMN at both the Fz and Cz sites. No significant differences in MMN were found between the groups (Table 3).

4. Discussion

This is the first study to investigate sensory processing deficiencies that may contribute to the origin of auditory verbal hallucinations (AVH) in patients with borderline personality disorder (BPD). Following the extensive number of studies on auditory hallucinations in patients with schizophrenia, we hypothesized that P50 sensory gating (measured as the P50 ratio and P50 difference) and mismatch negativity (MMN) would be negatively affected in patients with BPD and AVH, compared to patients with BPD without AVH and to healthy controls. Indeed, the P50 difference was significantly smaller in patients with BPD and AVH compared to healthy controls, with a moderate effect size ($r = 0.36$). Patients with BPD without AVH did not differ from healthy controls with regard to the P50 difference. Also, there was no significant difference between the three groups in P50 amplitudes, P50 ratio, and MMN.

The P50 difference is a more sensitive measurement of P50 sensory gating than the P50 ratio (Anokhin et al., 2007; Greenwood et al., 2007; Smith et al., 1994). This is probably why, in the present study, P50 difference showed a deficiency in patients with BPD and AVH whereas P50 ratio did not.

P50 sensory gating deficiency can be caused by either a defect in gating-out the test (S2) stimulus or a defective response to the conditioning (S1) stimulus (Chang et al., 2011). Chang, Arfken, Sangal & Boutros found, through weighted random effects meta-analysis, that the major contributor to sensory gating deficiencies is the decreased P50 amplitude elicited by *S2* (effect size 0.65, compared to 0.19 for the increased P50 amplitude elicited by S1) in studies comparing patients with schizophrenia and healthy controls (Chang et al., 2011). At the same time, they demonstrate a large range in P50 amplitudes in response to *S1* and a high percentage of overlapping between patients with schizophrenia and healthy controls. The authors therefore do not rule out a contribution of P50 amplitude elicited by S1 to sensory gating deficiencies. This 'gating-in' deficit has been described in patients with schizophrenia (Greenwood et al., 2016; Johannesen et al., 2005; Smith et al., 2013; Zhu et al., 2017; Zouridakis et al., 1997) and schizotypal personality disorder (Hazlett et al., 2015). Correspondingly, we found that P50 sensory gating deficiency in patients with BPD and AVH seems

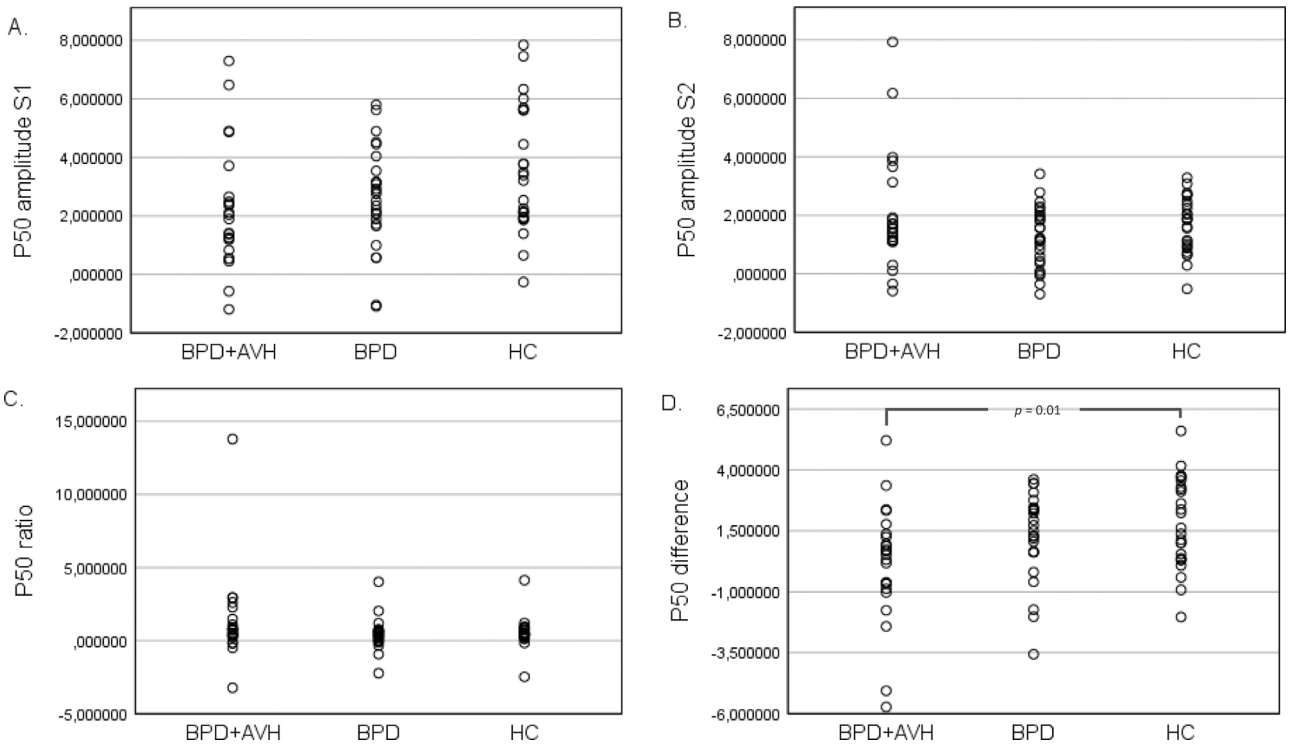

Fig. 2. Scatterplots for the P50 amplitude. The upper two graphs show the P50 amplitude elicited by (A) S1 and by (B) S2. The lower two graphs show the P50 ratio (C; S2/S1) and the P50 difference (D; S1-S2). Please note the use of different scaling. P-values are given of significant differences ($p < 0.05$) after Bonferroni correction for multiple comparisons. Abbreviations: BPD + AVH = patients with borderline personality disorder *and* auditory verbal hallucinations, BPD = patients with borderline personality disorder *without* auditory verbal hallucinations, HC = healthy controls, S1 = first stimulus, S2 = second stimulus.

Table 3
Data on mismatch negativity (MMN): mean (standard deviation) and test statistic.

	Mean (SD)			*F*	*p*
	1. BPD + AVH (*n* = 21)	**2. BPD (*n* = 25)**	**3. HC (*n* = 25)**		
MMN					
At Fz-site (μV)	−3.58 (2.51)	−4.57 (2.62)	−4.10 (1.95)	1.003	0.372
At Cz-site (μV)	−3.63 (2.61)	−4.86 (2.63)	−4.03 (2.22)	1.488	0.233

Abbreviations: MMN = mismatch negativity, BPD + AVH = patients with borderline personality disorder *and* auditory verbal hallucinations, BPD = patients with borderline personality disorder *without* auditory verbal hallucinations, HC = healthy controls.

to be caused by a diminished response to the S1 stimulus rather than to the S2 stimulus.

Our finding of a relationship between P50 sensory gating deficiency and AVH was demonstrated in three earlier studies (Faugere et al., 2016; Smith et al., 2013; Thoma et al., 2017) of patients with schizophrenia. Several other studies however, could not establish a correlation (Hamilton et al., 2018b) (and for a review see (Potter et al., 2006)). These studies show that associations with cognitive performance, more specifically attention, working memory and processing speed seem to be more robust than associations with AVH. In a short review, Ford mentioned a number of possible explanations for the lack of a significant association between ERP's, including P50, and the presence of positive or negative psychotic symptoms (Ford, 2018). For example, the use of psychotropic medication may alter the outward presentation (such as hallucinations) of the underlying pathology, leading to a decoupling of the symptoms from neurobiology. Ford also referred to the RDoC (Research Domain Criteria) framework of the National Institute for Mental Health (NIMH) which aims to cut across traditional disorder boundaries and focus on basic dimensions of functioning instead (Cuthbert and Insel, 2010). ERP's may be more beneficial in discovering the pathophysiology of psychotic symptoms when studied in relation to the specific domains of functioning that *underlie* the symptoms of interest (e.g. the Cognitive Systems Domain, encompassing among others attention and working memory) instead of in relation the specific symptom itself.

The fact that we did find P50 sensory gating deficiency in the BPD group who experienced AVH, as was found before in patients with schizophrenia (de Wilde et al., 2007b; Earls et al., 2016; Patterson et al., 2008) and patients with other diagnoses that display psychotic symptoms such as bipolar disorder and schizotypal disorder (Cabranes et al., 2013; Cadenhead et al., 2000, 2002; Olincy and Martin, 2005; Sanchez-Morla et al., 2008; Schulze et al., 2007), may imply that P50 sensory gating not only serves as a specific endophenotype for schizophrenia, but rather as an indication of psychotic vulnerability across diagnoses. We therefore recommend using a transdiagnostic symptomatic approach in the research, diagnosis and treatment of AVH (Javitt, 2016; Waters and Fernyhough, 2017).

In their review on the investigation of BPD via electrophysiological modalities, Boutros, Torella & McGlashan mention a number of studies that bring up the possibility of biologically distinct subtypes; i.e. an affective and a psychotic subtype, that may be useful in guiding treatment choices (Boutros et al., 2003). Future studies using biological markers such as ERPs could be useful to disentangle specific subtypes based on sensory processing.

In contrast to the present study, Grootens et al. found that patients with BPD showed *increased* sensory gating (P50 difference) compared to healthy controls, where we found *no* sensory gating differences between patients with BPD without AVH and healthy controls (Grootens et al., 2008). It seems Grootens et al. included a relatively healthy group of patients with BPD, since all comorbid mood disorders with the exception of dysthymic disorder were excluded, while these disorders are

known to be highly prevalent among patients with BPD (Zanarini et al., 2004).

Mismatch negativity was not affected in patients with BPD with or without AVH. This finding is comparable to that of He et al. who also found that MMN amplitudes in patients with BPD were not different from those in healthy controls (He et al., 2010).

The absence of MMN deficiency in patients with BPD and AVH may be explained by our choice to use a *frequency* deviant stimulus to investigate change detection in auditory stimulation. A study by Michie et al., investigating reasons why a minority of studies did not show MMN deficiency in patients with schizophrenia, showed that only duration-deviant stimuli led to significantly reduced MMN, while frequency-deviant stimuli MMN showed a trend in reducing MMN, provided that large frequency deviants were used (Michie et al., 2000).

There is also another possible explanation for the absence of MMN deficiency in patients with BPD and AVH. Although attenuated MMN has been established in patients with chronic schizophrenia (Erickson et al., 2016; Naatanen et al., 2014), equivocal results (with also intact or minimally deviant MMN) were found in ultra-high-risk and first-episode schizophrenia patients. In healthy voice hearers MMN deficiencies have even been demonstrated to be absent (van Lutterveld et al., 2010). Additionally, MMN deficiency was found to be specific for the patients with schizophrenia that are significantly disabled and unable to function with or without support (Hamilton et al., 2018a). Thus, it seems that, rather than MMN deficiency serving as a genetic vulnerability for the disease (schizophrenia), it might be better seen as a marker for disease progression and poor outcome (Erickson et al., 2016; Hamilton et al., 2018a; Umbricht and Krljes, 2005) in individuals who experience psychotic symptoms. If we translate this hypothesis to our group of patients with BPD and AVH in whom MMN deficiencies were absent, this group (as are the healthy voice hearers) appears to be spared from the poor outcome associated with negative psychotic symptoms and symptoms of disorganization seen in patients with chronic schizophrenia. This seems to be in line with the findings in our previous study, demonstrating the absence of negative symptoms and disorganization in patients with BPD and AVH, thereby distinguishing them from patients with schizophrenia (Niemantsverdriet et al., 2017).

4.1. Study limitations

The significance of the difference between patients with BPD and AVH and BPD without AVH with regards to P50 difference, disappeared after Bonferroni correction. We can not rule out that this means that these two groups do not differ in this regard and therefore the significance for P50 difference between patients with BPD and AVH and healthy controls is not explained by the presence of AVH. However, observing the absolute median values for P50 difference (BPD and AVH 0.53 μV, BPD and healthy controls both 1.62 μV) does not make this assumption very likely. Rather, we expect that the relatively small sample sizes have prevented the results to reach statistical significance. The sample sizes were based on an expected large effect size (Bramon et al., 2004), instead a moderate effect size was found for P50 difference between patients with BPD and AVH and healthy controls.

The reduced P50 amplitude to the S1 stimulus for patients with BPD and AVH may have confounded the finding of the smaller P50 difference, as the reduced P50 amplitude to the S1 stimulus creates a floor effect for the P50 amplitude to the S2 stimulus for the patients with BPD and AVH in contrast with the healthy controls for whom there is no floor effect. This limitation warrants caution to draw definite conclusions with regards to P50 sensory gating deficits in patients with BPD and AVH until replications of the results with larger sample sizes are established.

It could be argued that, in the present study, the use of psychotropic medication by participants with BPD both with and without AVH, may have influenced MMN thereby explaining the lack of differences compared with healthy controls. Participants in the present study were instructed not to take benzodiazepines on the day of the EEG and chronic use of benzodiazepines does not influence this ERP component (Kasai et al., 2002). In addition, no clear effects of antipsychotics on MMN have been demonstrated (Umbricht and Krljes, 2005). However, no studies have investigated the effect of mood stabilizers on MMN. Furthermore, one study found that escitalopram did enhance MMN in healthy participants (Wienberg et al., 2010). Therefore, an effect of mood stabilizers or antidepressants on MMN cannot be fully ruled out.

Also, as there may be a normalizing effect of smoking on P50 sensory gating and the effects of nicotine are short-lived, it is advised not to let participants smoke before testing (de Wilde et al., 2007b). We did not control for smoking and did not instruct our participants not to smoke before the testing. However, before testing, filling in the questionnaires and preparing the participant for the test took about 1–2 h, which may be long enough to allow any possible effects of nicotine to subside.

4.2. Recommendations for research

Replication studies are necessary to establish that sensory gating deficiencies are indeed a consistent finding in patients with BPD experiencing AVH. Since the sound intensity of the stimuli correlates with the effect size of P50 suppression (de Wilde et al., 2007a), we recommend that future studies use a standard stimulus of 15 dB above the hearing threshold (Griffith et al., 1995). In addition, increased sound intensities can be used to elicit larger P50 amplitudes. This might yield more distinctive results and provide more insight into whether P50 sensory gating deficiency in patients with BPD and AVH is caused by a 'gating-in' (a diminished response to the S1 stimulus) or a 'gating-out' (suppression of the response to the S2 stimulus) deficit. Also, a direct comparison with patients with schizophrenia might reveal whether P50 sensory gating is impaired to the same (or to a lesser) extent in patients with BPD and AVH. For example, less impairment was found in patients with schizotypal personality disorder, which the authors attributed to possible protective factors, such as frontal lobe sparing (Hazlett et al., 2015).

Furthermore, P50 sensory gating deficiency has been found in patients with post-traumatic stress disorder (PTSD) (Javanbakht et al., 2011; Karl et al., 2006) and is now also demonstrated in patients with BPD and AVH. Patients with BPD and AVH display a higher prevalence of PTSD than patients with BPD without AVH (Niemantsverdriet et al., 2017). These findings may imply that traumatic experiences are a causal factor in the development of P50 sensory gating deficiency leading to psychotic symptoms. Although this relationship between (childhood) trauma and the development of psychotic symptoms is widely acknowledged (McCarthy-Jones and Longden, 2015; Niemantsverdriet et al., 2017; Varese et al., 2012), the exact etiological routes remain to be elucidated.

In addition, we need to examine whether attention, working memory and processing speed (which are associated with P50 sensory gating deficiency in patients with schizophrenia) also play a role in the experience of AVH in patients with BPD, and whether interventions aimed at improving attention and working memory can improve P50 sensory gating and decrease the distress/dysfunction caused by AVH (Popov et al., 2011). Transcranial direct current stimulation (tDCS) is another possible treatment aimed at modulating P50 sensory gating to improve auditory hallucinations, which may be worth investigating when more definite results in patients with schizophrenia become available (Kim et al., 2018). Finally, in view of the positive effects of clozapine on P50 sensory gating (Adler et al., 2004; Becker et al., 2004; Nagamoto et al., 1999; Simosky et al., 2003), it is important to examine whether this effect can also be achieved in patients with BPD and AVH, and whether improved P50 sensory gating leads to diminishment of AVH in this patient group.

Declaration of competing interest

None. The authors report no potential conflicts of interest.

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Supplementary materials

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